03-13-08

AF/CIP

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of:

Beyaert et al.

Serial No.: 10/680,998

Filed: October 8, 2003

For: NOVEL INHIBITORS OF NF-kappaB ACTIVATION

Confirmation No.: 7433

Examiner: A. Rooke

Group Art Unit: 1656

Attorney Docket No.: 2676-4554.1US
(VAL/RBE/ABIN/cs/00-4253)

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BRIEF ON APPEAL

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P.O. Box 1450
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Attention: Board and Patent Appeals and Interferences

Sirs:

This Appeal Brief is submitted in the format required by 37 C.F.R. § 41.37 and with the fee required by 37 C.F.R. § 41.20(b) (2).

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I. REAL PARTY IN INTEREST

U.S. Application Serial No. 10/680,998 ("the '998 Application"), the application at issue, has been assigned to the Vlaams Interuniversitair Instituut voor Biotechnologie ("VIB"), as evidenced by the assignment via '998's parent application, Serial No. 09/702,953, now US Patent No. 6,673,897, that has been recorded with the U.S. Patent and Trademark Office ("USPTO") at Reel No. 011657, Frame No. 0476. Accordingly, the Vlaams Interuniversitair Instituut voor Biotechnologie is the real party of interest.

II. RELATED APPEALS AND INTERFERENCES

There are no appeals or interferences known to appellant or their representatives related to the pending appeal that will directly affect, be directly affected by, or otherwise have a bearing on the Board's decision in the pending appeal.

III. STATUS OF CLAIMS

Claims 19-24 are currently pending in the '998 Application. Claims 19, 20, and 24 are allowed. Claim 23 is objected to. Claims 21 and 22 stand rejected. Claims 1 through 18 were previously canceled without prejudice or disclaimer. The rejections of claim 21 and 22 are being appealed.

IV. STATUS OF AMENDMENTS

The appellants' amendments under 37 C.F.R. § 1.116, filed October 1, 2007, have been entered.

V. SUMMARY OF CLAIMED SUBJECT MATTER

In fulfillment of the provisions of 37 C.F.R. § 41.37(c)(1)(v), appellants provide a summary of the claimed subject matter for each of the independent claims at issue. The present application currently presents one independent claim that is at issue in the present appeal: independent claim 21.

Claim 21 provides for a method of screening a compound for its ability to activate or

suppress ABIN (A20-Binding Inhibitor of NF-κB activation) dependent NF-κB inhibition. The method comprises: a) combining a compound to be screened with a protein comprising ABIN amino acid consensus sequence of SEQ ID NO:9 and having the ability to inhibit NF-kB activation [described in the application as-filed in at least page 6, lines 1-3; page 22, lines 26-30; Example 5, page 23, lines 4-15; Example 6, page 24, lines 14-30]; b) detecting an interaction between said compound and said protein [*Id.* in at least Example 3, pages 21, lines 1-9 and 18-30]; c) identifying compounds that interact with said protein [*Id.* in at least page 6, lines 4-6; Example 3, pages 21, lines 1-9 and 18-30]; d) obtaining a cell line with a nucleic acid sequence encoding said ABIN consensus sequence protein and an NF-kB dependent reporter gene [*Id.* in at least page 10, lines 10-16; Example 3, pages 21, lines 1-9 and 18-30; Example 5, page 23, lines 4-15]; e) administering to the cell line a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein [*Id.* in at least Example 5, page 23, lines 16-20; Example 6, page 24, lines 14-30]; f) administering one of the detected compounds to said cell line [*Id.* in at least Example 5, page 23, lines 4-20; Example 6, page 24, lines 7-26]; and g) determining if the administration of the detected compound alters NF-kB dependent reporter gene expression, wherein an increase in expression indicates that the detected compound suppresses ABIN dependent NF-kB inhibition and a decrease in expression indicates that the detected compound activates ABIN dependent NF-kB inhibition [*Id.* in at least FIGs. 4, 6, and 7; Example 5, page 23, lines 4-30; page 24, lines 2-4].

As set forth in 37 C.F.R. 41.73 (c) (1) (vii), every means plus function claim must be identified and the structure, materials, or acts described in the specification corresponding to each claimed function must be set forth with reference to the specification. The current application contains a single means plus function claim, to wit: claim 21. The relevant means plus function language of claim 21 recites "administering to the cell line a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein."

The specification describes several means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein. The means, as disclosed herein, were acknowledged by the Examiner. *See,* Final Office Action, page 3. The disclosed means include: **TNF (tumor necrosis factor)** [described in the application as-filed in

at least FIGs. 4, 5, 7, 11, and 12; Page 5, lines 20-21 and 28-30; Page 6, lines 1-3; Page 15, lines 5-9 and 18-21; Page 16, lines 13-24; Example 5 at Page 23, lines 4-6, 21, and 29-30]; **IL-1 (interleukin-1)** [*Id.* in at least FIG. 4; Page 5, lines 20-21 and 28-30; Page 15, lines 5-9; Example 5 at Page 23, lines 4-6, 21, and 29-30]; **TPA (tissue plasminogen activator)**) [*Id.* in at least FIGs. 6 and 7; Page 15, lines 13-21; Example 5 at Page 23, lines 4-6 and 17-19; Page 24, lines 1-3]; **TRADD (TNF receptor associated death domain)** [*Id.* in at least FIGs. 8, 9; Page 15, lines 23-30; Example 6 at Page 24, lines 7-26]; **RIP (receptor interacting protein)** [*Id.* in at least FIGs. 8, 9; Page 15, lines 23-30; Example 6 at Page 24, lines 7-26]; and **TRAF2 (TNF receptor associated factor 2)** [*Id.* in at least FIGs. 8, 9; Page 15, lines 23-30; Page 21, lines 24-25; Example 6 at Page 24, lines 7-26].

VI. GROUNDS OF REJECTION TO BE REVIEWED ON APPEAL

(i) Whether claims 21 and 22 are unpatentable under 35 U.S.C. § 112, first paragraph, as not complying with the written description requirement?

(ii) Whether claims 21 and 22 are unpatentable under 35 U.S.C. § 112, first paragraph, as not complying with the enablement requirement?

VII. ARGUMENT

(i) Rejection of claims 21 and 22 under 35 U.S.C. § 112, first paragraph, written description requirement

Claims 21 and 22 were rejected under 35 U.S.C. § 112, first paragraph, as allegedly failing to comply with the written description requirement. The rejection stated that Claim 21 included subject matter that was allegedly not described in the Specification in such a way as to reasonably convey to one skilled in the art that the inventors, at the time the Application was filed, had possession of the claimed invention. *See,* Final Office Action, page 3. Specifically, the Examiner alleges that "applicants did not adequately disclose 'a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein.'" *Id.* To support this allegation, the Examiner asserts that "there can be many

different structures/chemicals/factors, other than those disclosed... that can achieve the same effect and purpose in the method claimed." *Id.*

Appellants respectfully submit that the Examiner failed to properly examine the element at issue in claims 21 (namely, "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein") in light of 35 U.S.C. §112, paragraph 6. Appellants further submit that claim 21 complies with the written description requirement, because (I) the element of claim 21 at issue in the Examiner's rejection (namely, "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein") is a "means-plus-function element" entitled to examination in light of 35 U.S.C. § 112, paragraph 6, and (II) the element at issue in claim 21 satisfies the two-part test established by the United States Patent and Trademark Office to establish compliance with the written description requirement by means-plus-function elements.

I. *"A means for inducing activation of the NF-κB pathway..." should be examined in light of 35 U.S.C. § 112, paragraph 6.*

Congress has allowed, by statute, for patent applicants to claim subject matter according to a means for performing a specified function. 35 U.S.C. § 112, paragraph six, states:

> An element in a claim for a combination may be expressed as a means or step for performing a specified function without the recital of structure, material, or acts in support thereof, and such claim shall be construed to cover the corresponding structure, material, or acts described in the specification and equivalents thereof. 35 U.S.C. § 112, ¶ 6.

In determining whether claim elements should be examined under the "means-plus-function" language as directed by 35 U.S.C. § 112, paragraph 6, the United States Patent Office has established a test. That test, as provided in MPEP 2181(I), is:

> A claim limitation will be presumed to invoke 35 U.S.C. 112, sixth paragraph, if it meets the following 3-prong analysis: (A) the claim limitations must use the phrase "means for" or "step for;" (B) the "means for" or "step for" must be modified by functional language; and (C) the phrase "means for" or "step for" *must not be modified by sufficient structure, material, or acts for achieving the specified function.* Emphasis added for clarity. MPEP 2181(I).

In the '998 Application, adequate written description and disclosure of the element at issue in claim 21 should be examined in light of 35 U.S.C. § 112, paragraph 6, because the means-plus-function element of claim 21 clearly satisfies the 3-prong analysis set forth by the Patent Office. First, the relevant portion of claim 21 recites "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein..." The element at issue recites the phrase "means for" or "step for" as dictated by the first prong (A) of the USPTO's own analysis.

Similarly, the means-plus-function element in claim 21 clearly satisfies the second prong (B) of the analysis, which states that, the "means for" or "step for" must be modified by functional language. The means-plus-function element recites "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein..." Thus, the "means for" in claim 21 is modified by the functional language "inducing activation of the NF-κB pathway", and is further modified by the functional language "wherein the means is inhibitable by said ABIN consensus sequence protein." Accordingly, the "means for" in claim 21 is modified by functional language.

The third prong (C) of the PTO's analysis is also satisfied with regard to the means-plus-function element in claim 21. The third prong states that the phrase "means for" or "step for" must not be modified by sufficient structure, material, or acts for achieving the specified function. *See,* MPEP 2181(I). No where in "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein" is there any recitation of structure, material, or acts for achieving the specified function, namely, inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein.

Appellants respectfully submit that the means-plus-function element in claim 21, "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein" clearly satisfies the three prongs as delineated in MPEP 2181(I). Thus, the USPTO's own guidelines, both statutory and procedural, demonstrate the means-plus-function element in claim 21 should be examined in light of 35 U.S.C. § 112, paragraph 6.

Appellants additionally respectfully submit that the Examiner failed to properly examine the written description and disclosure of this element of claim 21 in light of 35 U.S.C. §112, paragraph 6. As noted previously, 35 U.S.C. §112, paragraph 6, states that if an element of a claim is expressed in terms of means plus function, "such claim shall be construed to cover the corresponding structure, material, or acts described in the specification and equivalents thereof." 35 U.S.C. § 112, ¶ 6. In both the Office Action of June 1, 2007 and the Final Office Action of December 28, 2007, the Examiner did not properly construe the scope of the Claim 21 in light of §112, paragraph 6.

Appellants respectfully note that 35 U.S.C. §112, paragraph 6, mandates the scope of and interpretation of claims that include means-plus-function elements. *See, In re Donaldson,* 16 F.3d 1189, 1193; 29 USPQ 2d 1845, 1851 (Fed. Cir., 1994). Indeed, in "construing means-plus-function language in a claim one must look to the specification and interpret that language in light of corresponding structure, material, or acts describer therein, or equivalents thereof." *Id.* (emphasis added).

II. ***Claims 21 and 22 satisfy the written description requirement under the test established for examination of means-plus-function claims.***

Once a claim is entitled to examination as a means-plus-function claim, it still must be analyzed "to determine whether there exists corresponding adequate support for such claim under 35 U.S.C. 112, paragraph 1. MPEP 2181(IV).

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention. 35 U.S.C. § 112, first paragraph.
>
> If a claim limitation invokes 35 U.S.C. **112**, para. 6, it must be interpreted to cover the corresponding structure, materials, or acts in the specification and "equivalents thereof." See 35 U.S.C. **112**, para. 6. See also *B. Braun Medical, Inc. v. Abbott Lab.*, 124 F.3d 1419, 1424, 43 USPQ2d 1896, 1899 (Fed. Cir. 1997). A means- (or step-) plus-function claim limitation is adequately described under 35 U.S.C. 112, para. 1, if: (1) The written description adequately links or associates

> adequately described particular structure, material, or acts to the function recited in a means- (or step-) plus-function claim limitation; or (2) it is clear based on the facts of the application that one skilled in the art would have known what structure, material, or acts perform the function recited in a means- (or step-) plus-function limitation. MPEP 2163 II(A)3a.

Appellants respectfully submit that the '899 Specification and the evidence of record clearly demonstrate the compliance of claim 21 with the written description requirement of 35 U.S.C. §112, paragraph 1. In accordance with the MPEP, the '899 Specification adequately links or associates adequately the particular structures, materials, or acts (namely, TNF, IL-1, TPA, RIP, TRAF2) to the function recited in the means-plus-function claim element (the function being 'inducing activation of the NF-κB pathway' and 'means is inhibitable by said ABIN consensus sequence protein'). Further, the evidence of record (*e.g.*, applicants' declaration under 37 C.F.R § 1.132 and the Examiner's acknowledgments throughout prosecution) supports a finding of compliance with 35 U.S.C. §112, paragraph 1.

Appellants submit that the '899 Specification clearly and distinctly associates TNF, IL-1, TPA, RIP, TRAF2 ('means') with the function of "inducing activation of the NF-κB pathway, wherein the means are inhibitable by the ABIN consensus sequence protein." For example, Examples 5 and 6 state and demonstrate that TNF, IL-1, TPA, TRADD, RIP, TRAF2, etc. induce activation of the NF-κB pathway. *See, e.g.,* Paragraphs [0108]-[0112] and [0113]-[0116]. The experimental results from Examples 5 and 6 are further shown in FIGs. 4 through 9, indicating that TNF, IL-1, TPA, TRADD, RIP, TRAF2, etc. are capable of inducing activation of the NF-κB pathway.

Examples 5 and 6 additionally demonstrate that the 'means' (*e.g.,* TNF, IL-1, TPA, TRADD, RIP, TRAF2, etc.) are inhibitable by the ABIN consensus sequence protein. Example 5 states that both splice variants of ABIN and ABIN variants (referring to SEQ ID NOs 8 and 9, as described in Paragraph [0107]) were able to block TNF, IL-1, or TPA induced NF-κB activation. *Id.* at [0107] and [0110]. Example 6 found similar findings with respect to TRADD, RIP, and TRAF2. *Id.* at [0115].

During prosecution, the appellants further demonstrated claim 21's compliance with the written description requirement by submitting a Declaration under 37 C.F.R. § 1.132. The

Declaration provides evidence that one skilled in the art would know "what materials disclosed in the patent application (*e.g.,* TNF, IL-1, TPA, RIP, and TRAF2) would perform the function of 'a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein.'" *See,* Dec. under 37 CFR § 1.132, at paragraph 8 (submitted October 10, 2007). The Declaration further states that one skilled in the art would find adequate written description of the claimed means. *Id.* at 9.

Additionally, the Examiner's acknowledgement of compliance of the disclosed 'means' provides ample evidence that the disclosed means comply with the written description requirement of 35 U.S.C. §112, paragraph 1. Indeed, the Examiner acknowledges that TNF, IL-1, TPA, RIP, and TRAF2 are adequately disclosed means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by the ABIN consensus sequence protein. *See,* Final Office Action at 6; Office Action mailed June 1, 2007 at Pages 3 (see allowable claim 19) and 7 (see Objection to claim 23, objected to only because of dependency to claim 21, and which remedies the asserted lack of written description of claim 21 by specifically reciting TNF, IL-1, TPA, RIP, and TRAF2).

The Examiner responded to his own acknowledgements by stating that "the adequate disclosure was in reference to the allowed claims 19, 20, 23, and 24" and not to claims 21 and 22. *See,* Final Office Action, page 6. However, as argued by the appellants, an acknowledgement of adequate disclosure of Claim 23 is, at the very least, an acknowledgement of the adequate disclosure of the 'means' as disclosed in claims 21 and 22. Indeed, claim 23 which currently stands objected to for the sole reason its dependency on claim 21, includes a list of the 'means' at issue, namely TNF, IL-1, TPA, RIP, TRAF2.

In rejecting claim 21, the Examiner alleged, that "because there can be many different structures/chemicals/factors other than disclosed TNF, IL-1, TPA, RIP, TRAF2, that can achieve the same effect and purpose in the method claimed, the appellants are not entitled to all of these undisclosed means of inducing activation." *See,* Final Office Action at 5; Office Action mailed June 1, 2007 at Page 5. However, the Examiner's assertion regarding the number of undisclosed means of inducing activation is in direct contravention to 35 U.S.C. § 112, paragraph 6. As noted above, the MPEP and 35 U.S.C. § 112, paragraph 6, provide, in part, that "such claims

shall be construed to cover the corresponding structure, material, or acts described in the specification and equivalents thereof." As such, 35 U.S.C. § 112, paragraph 6, dictates that the "means" must be construed to cover the corresponding structure, material, or acts described in the specification and equivalents thereof.

Additionally, in response to appellants' arguments, the Examiner stated that "the rejected claims 21 and 22 do not point out a single compound that induces activation of the NF-κB pathway." Final Office Action at page 7. Again, appellants point to the language of § 112, paragraph 6, where it states, "an element in a claim for a combination may be expressed as a means or step for performing a specified function without the recital of structure, material, or acts in support thereof..." 35 U.S.C. § 112, ¶ 6. Further, "such claim shall be construed to cover the corresponding structure, material, or acts described in the specification and equivalents thereof." *Id.* Accordingly, the statutory language dictates that means for elements, such as those in claims 21 and 22, need not recite structure, material, etc., and that such claim shall be construed to cover that which is disclosed in the Specification. Further, as previous noted herein, the Examiner has acknowledged that such structure and materials have been adequately described.

In view of at least the foregoing, claim 21 must be considered under 35 U.S.C. § 112, paragraph 6, and appellants respectfully submit that the Specification provides adequate written description of "a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein." Consequently, appellants respectfully request withdrawal of the rejection of claim 21 under 35 U.S.C., first paragraph, and reconsideration of same.

In addition, appellants respectfully submit that adequate written description exists for claim 22, *inter alia*, as claim 22 depends from adequately described claim 21. Consequently, appellants respectfully request withdrawal of the rejection of claim 22 under 35 U.S.C., first paragraph, and reconsideration of same.

(ii) Rejection of claims 21 and 22 under 35 U.S.C. § 112, first paragraph, enablement requirement

Claims 21 and 22 were rejected under 35 U.S.C. § 112, first paragraph as allegedly failing

to comply with the enablement requirement. Specifically, the Examiner alleged that the Specification, while being enabling for a method of screening a compound for its ability to activate or express ABIN when TNF, IL-1, TPA, RIP, TRAF2 are administered to the cell line to induce activation of the NF-κB pathway, "[the specification] does not reasonably provide enablement for administering to the cell line any means for inducing activation of the NF-κB pathway." *See,* Final Office Action, page 3, emphasis by Examiner.

Appellants respectfully submit that, similar to the Examiner's previous rejection, the Examiner failed to properly examine the means-plus-function element in claim 21 (namely, "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein") in light of 35 U.S.C. §112, paragraph 6. Appellants further submit that claim 21 complies with the enablement requirement, because (I) the element of claim 21 at issue in the Examiner's rejection (namely, "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein") is a "means-plus-function" element entitled to examination under 35 U.S.C. § 112, paragraph 6, and (II) the means-plus-function element in claim 21 satisfies the test established by the United States Patent and Trademark Office to establish compliance with the enablement requirement by means-plus-function claims.

I. ***"A means for inducing activation of the NF-κB pathway..." should be examined in light of 35 U.S.C. § 112, sixth paragraph.***

With reference to the appellants' arguments from the previous rejection, appellants respectfully submit that the element at issue in claim 21, namely "a means for inducing activation of the NF-κB pathway, wherein the means is inhibitable by the ABIN consensus sequence protein," clearly satisfies the three-prongs test as delineated in MPEP 2181(I). Thus, the USPTO's own guidelines demonstrate the element at issue is a means-plus-function element and therefore, should be examined in light of 35 U.S.C. § 112, paragraph 6.

Similar to the previous rejection, appellants additionally submit that the Examiner failed to properly examine the enablement of claim 21 in light of 35 U.S.C. §112, paragraph 6. In both the Office Action of June 1, 2007 and the Final Office Action of December 28, 2007 the

Examiner alleges that "[the specification] does not reasonably provide enablement for administering to the cell line any means for inducing activation of the NF-κB pathway." *See,* Final Office Action, page 3.

As noted previously, the scope of the means-plus-function element in claims 21 and 22 is mandated according to 35 U.S.C. §112, paragraph six, that scope being that which is described in the specification and equivalents thereof. Thus, in determining enablement under 35 U.S.C. § 112, the proper scope is not "any means" as alleged by the Examiner, but rather the structures, materials, etc. described in the Specification, and equivalents thereof.

II. ***Claims 21 and 22 satisfy the enablement requirement***

Appellants respectfully submit that the Specification and the evidence of record clearly demonstrate the compliance of claim 21 with the enablement requirement of 35 U.S.C. § 112, paragraph 1.

Appellants note that the Court of Appeals for the Federal Circuit (CAFC) has held that with regard to means-plus-function elements, "there is and can be no requirement that applicants describe or predict every possible means of accomplishing" the function set forth in the means-plus-function element. *See, D.M.I. Inc. v. Deere & Co.,* 7755 F.2d 1570, 1574; 225 USPQ 236, 238 (Fed. Cir. 1985). Rather, the CAFC stated that patentees are required to disclose "some enabling means" for accomplishing the function set forth in the means-plus-function element.

First, the Specification clearly discloses several examples of the 'means' (*e.g.,* TNF, IL-1, TPA, RIP, TRAF2) that accomplish the function set forth in the means-plus-function element at issue in claim 21. Indeed, the Examiner acknowledged such enablement with respect to the means disclosed in claim 21. The Final Office Action states, "while being enabling for a method of screening a compound for its ability to activate or express ABIN when TNF, IL-1, TPA, RIP, TRAF2 are administered to the cell line to induce activation of the NF-κB pathway..." Final Office Action at 3. Additionally, the presently allowed and/or objected to claims provide evidence of enablement. *See,* Final Office Action at 6; Office Action mailed June 1, 2007 at Pages 3 (see allowable claim 19) and 7 (see objection to claim 23, which remedies the asserted

lack of written description of claim 21 by specifically reciting TNF, IL-1, TPA, RIP, and TRAF2).

Additionally, appellants note that "a claim is enabled under 35 U.S.C. § 112, paragraph 1, if an application, when filed, contains sufficient information regarding the subject matter of the claim as to enable one skilled in the pertinent art to make and use the claimed invention without undue experimentation. MPEP 2164.01; Mineral Separation v. Hyde, 242 U.S. 261, 270 (1916); In re Wands, 858 F.2d 731, 737 (Fed. Cir. 1988).

In light of the above, appellants submit that claim 21 is enabled under 35 U.S.C. § 112 paragraph 1, because the '899 Application, when filed, contained sufficient information regarding the subject matter of the claims as to enable one skilled in the pertinent art to make and use the claimed invention without undue experimentation. MPEP 2164.01; Mineral Separation v. Hyde, 242 U.S. 261, 270 (1916); In re Wands, 858 F.2d 731, 737 (Fed. Cir. 1988).

In addition, appellants respectfully submit that adequate enablement exists for claim 22, *inter alia*, as claim 22 depends from adequately enabled claim 21. Consequently, appellants respectfully request withdrawal of the rejection of claim 22 under 35 U.S.C., first paragraph, and reconsideration of same.

VIII. CLAIMS APPENDIX

1.-18. (Canceled).

19. (Previously Presented) A method of screening a compound for its ability to activate or suppress ABIN (A20-Binding Inhibitor of NF-κB activation) dependent NF-κB inhibition, said method comprising:

a) combining a compound to be screened with a protein comprising ABIN amino acid consensus sequence of SEQ ID NO: 9 and having the ability to interact with protein A20,

b) detecting an interaction between said compound and said protein,

c) identifying compounds that interact with said protein,

d) obtaining a cell line with that nucleic acid sequence encoding protein A20, nucleic acid sequence encoding said ABIN consensus sequence protein, and an NF-kB dependent reporter gene,

e) administering at least one of TNF (tumor necrosis factor), IL-1 (interleukin-1), TPA (tissue plasminogen activator), TRADD (TNF receptor associated death domain), RIP (receptor interacting protein), TRAF2 (TNF receptor associated factor 2) to the cell line to induce activation of the NF-kB pathway,

f) administering the detected compounds to said cell line, and

g) determining if the administration of the detected compounds alter NF-kB dependent reporter gene expression, wherein an increase in expression indicates that the detected compounds suppress ABIN dependent NF-kB inhibition and a decrease in expression indicates that the compounds activate ABIN dependent NF-kB inhibition.

20. (Previously Presented) The method according to claim 19, wherein detecting an interaction between said compound and said protein comprises using either a two-hybrid assay or a co-immunoprecipitation assay.

21. (Previously Presented) A method of screening a compound for its ability to activate or suppress ABIN (A20-Binding Inhibitor of NF-kB activation) dependent NF-kB inhibition, said method comprising:

a) combining a compound to be screened with a protein comprising ABIN amino acid consensus sequence of SEQ ID NO:9 and having the ability to inhibit NF-kB activation,

b) detecting an interaction between said compound and said protein,

c) identifying compounds that interact with said protein,

d) obtaining a cell line with a nucleic acid sequence encoding said ABIN consensus sequence protein and an NF-kB dependent reporter gene,

e) administering to the cell line a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein,

f) administering one of the detected compounds to said cell line, and

g) determining if the administration of the detected compound alters NF-kB dependent reporter gene expression, wherein an increase in expression indicates that the detected compound suppresses ABIN dependent NF-kB inhibition and a decrease in expression indicates that the detected compound activates ABIN dependent NF-kB inhibition.

22. (Previously Presented) The method according to claim 21, wherein obtaining a cell line comprises obtaining a cell line including a nucleic acid sequence encoding protein A20.

23. (Previously Presented) The method according to claim 21, wherein the means for inducing activation of the NF-kB pathway comprises at least one of TNF (tumor necrosis factor), IL-1 (interleukin-1), TPA (tissue plasminogen activator), TRADD (TNF receptor associated death domain), RIP (receptor interacting protein), or TRAF2 (TNF receptor associated factor 2).

24. (Previously Presented) A method of screening a compound for its ability to activate or suppress ABIN (A20-Binding Inhibitor of NF-kB activation) dependent NF-kB inhibition, said method comprising:

a) combining a compound to be screened with a protein comprising ABIN amino acid consensus sequence of SEQ ID NO:9 and having the ability to inhibit NF-kB activation,

b) detecting an interaction between said compound and said protein,

c) identifying compounds that interact with said protein,

d) obtaining a cell line with a nucleic acid sequence encoding said ABIN consensus sequence protein, an NF-kB dependent reporter gene, and a NF-kB pathway inducible by at least one of TNF (tumor necrosis factor), IL-1 (interleukin-1), TPA (tissue plasminogen activator), TRADD (TNF receptor associated death domain), RIP (receptor interacting protein), or TRAF2 (TNF receptor associated factor 2),

e) inducing activation of the NF-kB pathway of said cell line,

f) administering one of the detected compounds to said cell line, and

g) determining if the administration of the detected compound alters NF-kB dependent reporter gene expression, wherein an increase in expression indicates that the detected compound suppresses ABIN dependent NF-kB inhibition and a decrease in expression indicates that the detected compound activates ABIN dependent NF-kB inhibition.

IX. EVIDENCE APPENDIX

A) Declaration under 37 C.F.R. § 1.132 of Dr. Rudi Beyaert, submitted October 10, 2007. Admitted and considered by Examiner Rooke as evidenced by Final Office Action of December 28, 2007, at page 7.

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of:

Beyaert et al.

Serial No.: 10/680,998

Filed: October 8, 2003

For: NOVEL INHIBITORS OF NF-kappaB ACTIVATION

Confirmation No.: 7433

Examiner: A. Rooke

Group Art Unit: 1656

Attorney Docket No.: 2676-4554.1US

DECLARATION UNDER 37 C.F.R. § 1.132 OF DR. RUDI BEYAERT

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Dr. Rudi Beyaert hereby declares:

1. I am a named inventor on the above-referenced patent application.

2. I am a Professor at the University of Ghent and an expert in the field of molecular signal transduction. A copy of my curriculum vitae is attached.

3. I understand that in the Office Action mailed June 1, 2007, the Examiner has rejected claims 21 and 22 as assertedly lacking written description and enablement

4. Submitted herewith are a published research paper (Wullaert *et al.*, J. Biol. Chem. 282:1 81-90, Jan. 5, 1997) and some additional data showing that Poly (I:C) (additional data), LPS (additional data and Wullaert *et al.* at FIG. 4B), BCL10 (additional data), API2-MLT (additional data), MyD88 (Wullaert *et al.* at FIG. 6), IRAK1 (Wullaert *et al.* at FIG. 6), TLR4

(Wullaert *et al.* at FIG. 4A), and TRAF6 (Wullaert *et al.* at FIG. 6) are all equivalents of TNF, IL-2, TPA, RIP, and TRAF2 in terms of each being a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by an ABIN consensus sequence protein.

5. I understand that claim 21 currently recites:

A method of screening a compound for its ability to activate or suppress ABIN (A20-Binding Inhibitor of NF-kB activation) dependent NF-kB inhibition, said method comprising:

a) combining a compound to be screened with a protein comprising ABIN amino acid consensus sequence of SEQ ID NO:9 and having the ability to inhibit NF-kB activation,

b) detecting an interaction between said compound and said protein,

c) identifying compounds that interact with said protein,

d) obtaining a cell line with a nucleic acid sequence encoding said ABIN consensus sequence protein and an NF-kB dependent reporter gene,

e) administering to the cell line a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by said ABIN consensus sequence protein,

f) administering one of the detected compounds to said cell line, and

g) determining if the administration of the detected compound alters NF-kB dependent reporter gene expression, wherein an increase in expression indicates that the detected compound suppresses ABIN dependent NF-kB inhibition and a decrease in expression indicates that the detected compound activates ABIN dependent NF-kB inhibition.

6. I understand that claim 22 currently recites:

The method according to claim 21, wherein obtaining a cell line comprises obtaining a cell line including a nucleic acid sequence encoding protein A20.

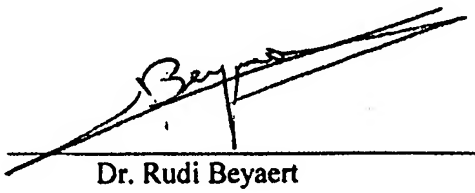
7. A person skilled in the art would know how to make and use the invention of claims 21 and 22 as TNF, IL-2, TPA, RIP, and TRAF2 are all described in the patent application in such a way as to indicate their ability to perform the function of "a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by an ABIN consensus sequence protein."

8. Such means are definite as one skilled in the art will understand what materials disclosed in the patent application (*e.g.* TNF, IL-1, TPA, RIP, and TRAF2) would perform the function of "a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by an ABIN consensus sequence protein."

9. A person skilled in the art would find adequate written description of a means for inducing activation of the NF-kB pathway, wherein the means is inhibitable by an ABIN consensus sequence protein as, *e.g.*, TNF, IL-1, TPA, RIP, and TRAF2 are described in the patent application as being capable of performing that function.

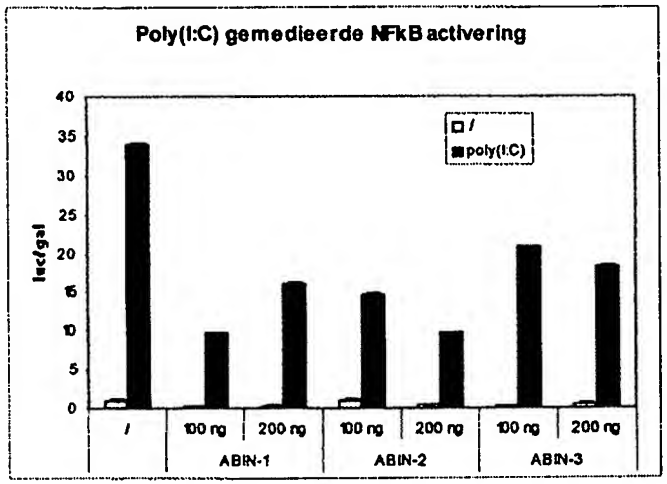
10. I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful false statements may jeopardize the validity of the application or any patents issued thereon.

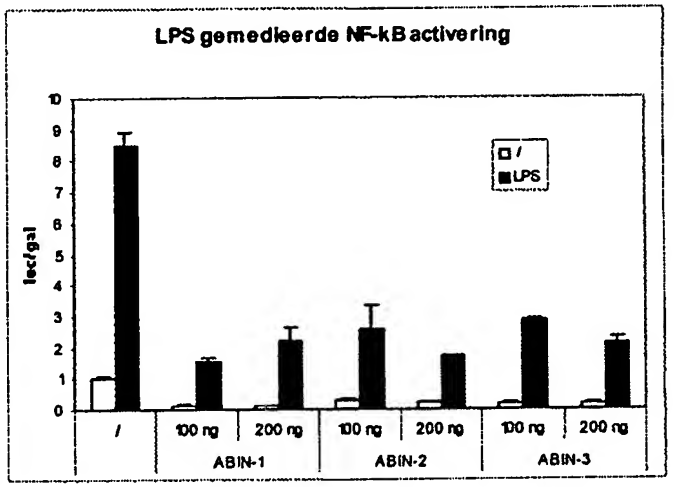
Date: Octobe, 1, 2007

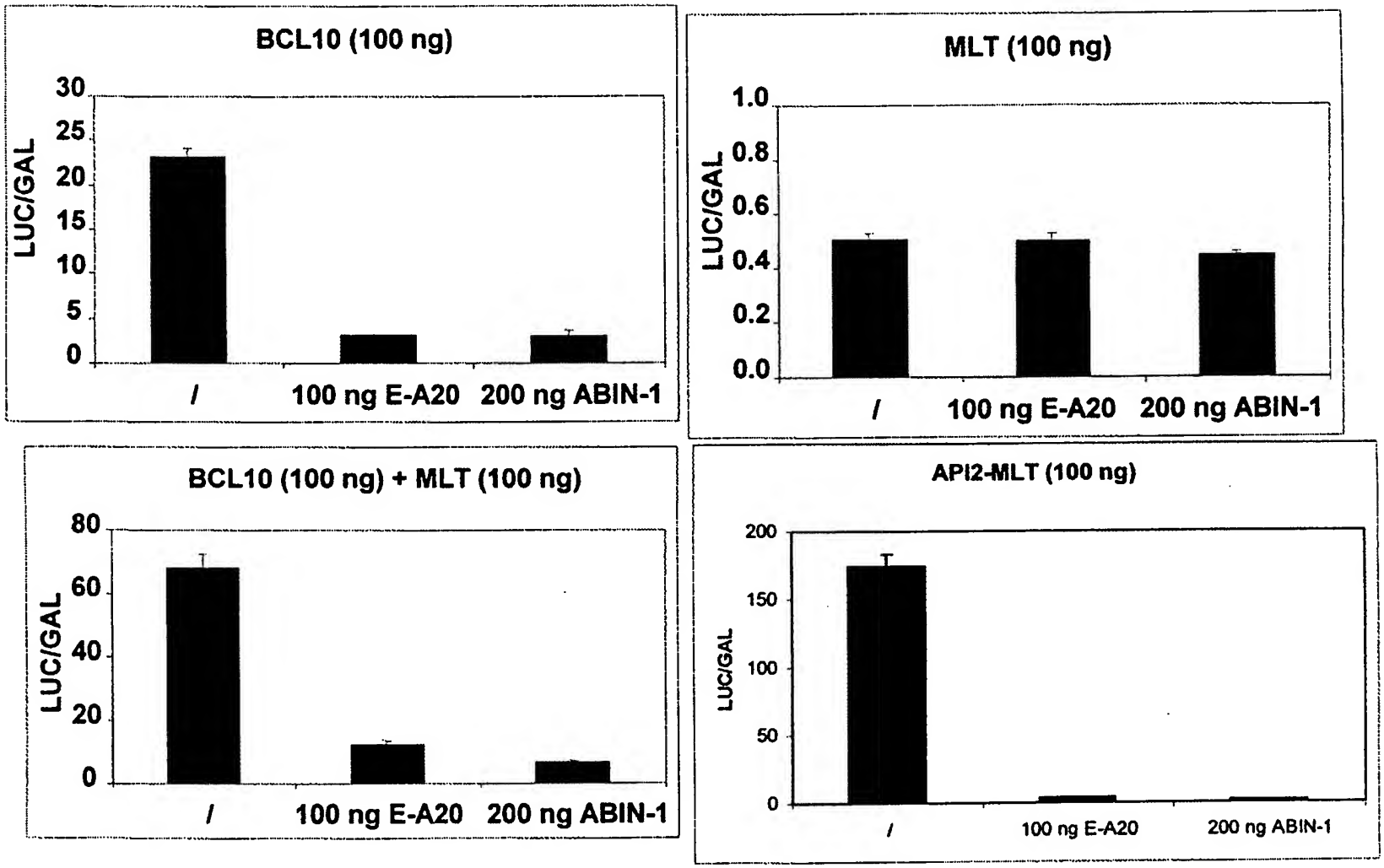


Dr. Rudi Beyaert

ABINs inhibit poly(I:C) and LPS induced NF-kB activation in respectively HEK-TLR3 and HEK-TLR4 cells







ABIN-1 inhibits Bcl10, Bcl10/MLT and API2/MLT induced NF-kB activation (= T cell and B cell receptor induced NF-kB activation)

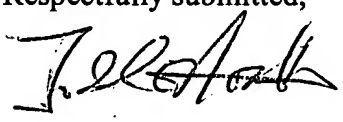
X. RELATED PROCEEDINGS APPENDIX

No decisions have been rendered by the Board or any court in a related application. Therefore, a related proceedings appendix does not accompany this Appeal Brief.

XI. CONCLUSION

Based on the foregoing arguments, appellants respectfully submit that claims 21 and 22 are in condition for immediate allowance. Consequently, appellants respectfully request that the rejection of claims 21 and 22 under 35 U.S.C. 112 , paragraph 1, for lack of written description the rejection of claims 21 and 22 under U.S.C. 112, paragraph 1, for lack of enablement be withdrawn, and the claims be allowed.

Respectfully submitted,



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Date: March 11, 2008
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If as part of this Appeal Brief, a Petition for Extension of Time should be required, please consider this document as the requisite Petition for Extension of Time. The Patent Office is authorized to charge TraskBritt Deposit Account 20-1469 in the amount of the required fee.

Respectfully submitted,

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